CRITICAL CARE NURSING COURSE
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Interpretation and Treatment of Cardiac Dysrhythmias Originating in the Ventricle

Objectives

1. Terminal Learning Objective

   Formulate a nursing treatment plan for the patient with cardiac dysrhythmias.

2. Enabling Learning Objectives

   a. Discuss the mechanisms which account for the genesis of cardiac dysrhythmias: disturbances of impulse formation,disturbances of impulse conduction; combined disturbances of impulse formation and conduction and fibrillation.

   b. Given the recommended format for interpreting dysrhythmias: identify the common dysrhythmias originating in the ventricles.

   c. Discuss the possible hemodynamic effects and treatment of the dysrhythmias.

   d. Discuss the nurse's role in continued assessment, monitoring and treatmentof each dysrhythmia.Objectives

NOTES

A. Review of Conduction - A special system concerned with transmission and coordination of electrical impulses

   1. Impulse formation - SA node primarily, however may develop from other inherent pacemakers in the A-V node and ventricles.

   2. Conduction pathways

      a. Once stimulated impulse transmitted via highly specialized network - when impulse reaches ventricles stimulation of the myocardium causes electrical depolarization of the cells and mechanical contraction

      b. Made up of SA node, internodal tracts, A-V node, Bundle of His, left and right bundle branches and Purkinje fibers

B. Ventricular Dysrhythmias

Originate in the ventricles below the branching of the bundle of His

C. Premature Ventricular Contractions (PVCs)

1. Not a rhythm itself; rather individual ventricular beats or a pair of ventricular ectopic beats which fire before the expected normal beat

2. Pediatrics: Occur in children with no history of heart disease. Benign

3. Mechanism

   a. Reentry through slowly conducting tissue in ventricle

   b. Enhanced normal automaticity in HIS-Purkinge system

   c. Abnormal automaticity in ventricle

   d. After depolarizations/tirggered activity

4. Causes - any substance or condition that increases ventricular irritability

   a. Electrical or chemical abnormalities (hypokalemia)

   b. Acute ischemia (with or without infarction)

   c. Hypoxia

   d. Fever

   e. Acidosis

   f. Myocardial stretch (CHF or ventricular dilatation)

   g. Injury - damaged cells leak sodium and potassium through their membrane

   h. Drugs - Digitialis excess or cathecholamines

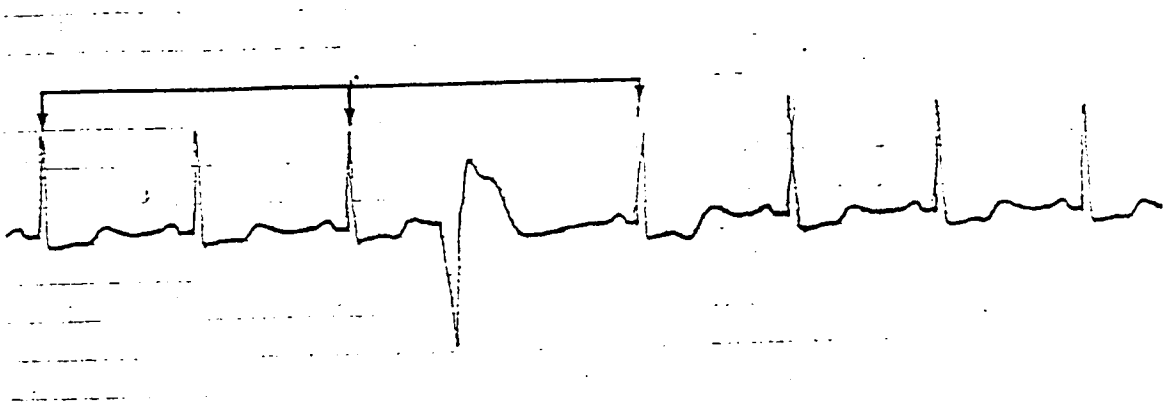
5. Clinical features

   a. Common

   b. Occur in people with and without heart disease

c. May increase with age, myocardial ischemia or infarct, hypertrophy, infection, increased sympathetic tone

Isolated Premature Ventricular Contraction



5. Significance

Reflects myocardial irritablility with MI→PVC's likely to provoke → ventricular fibrillation. Overdosages of digitalis, hypokalemia

6. ECG characteristics

a. Rate: Often normal; PVC arrives early

b. Rhythm: Irregular - caused by PVC and compensatory pause

c. P wave: Usually hidden in QRS complex, usually unrelated to QRS

d. QRS:

(1) Wider than 0.12 sec and usually wider than 0.14 sec

(2) Ventricular activation begins outside the conduction system and travels more slowly through the myocardium

(3) ST and T wave usually opposite direction from QRS

NOTE: Exceptions may include fascicular beats (originate high in bundle); fusion beats, or beats which may appear narrow in one lead.

NOTE: The strong vector of a PVC does not mean it is a "strong beat". It is, in fact, "weaker" as there is decreased diastolic filling time.

e. Compensatory pause: Follows a PVC and is caused by nonconducted normal sinus beat. 50% of the time there is retrograde AV block, if retrograde conduction occurs and a 'p' wave is usually there is no ventricular response because A-V node and ventricles are refractory. The next P wave occurs on time.

NOTE: A full compensatory pause proves ventricular ectopy only when nonconducted p wave can be seen.

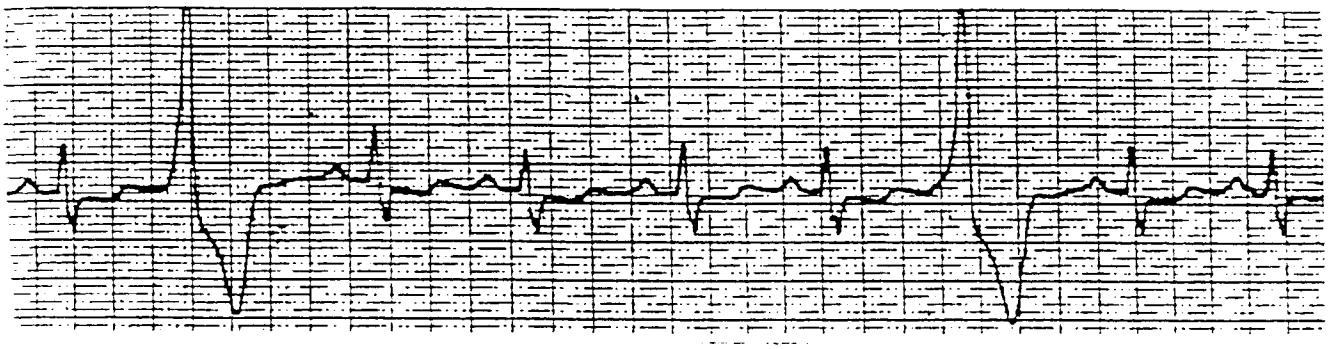
7. Types of PVCs

a. Unifocal

(1) Identical in shape

(2) Originate from same focus

(3) Current takes same route through myocardium

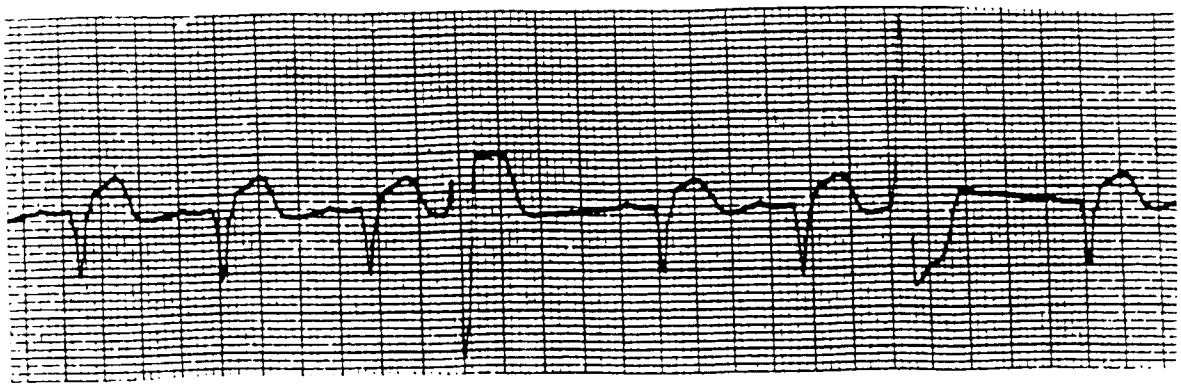


b. Multifocal

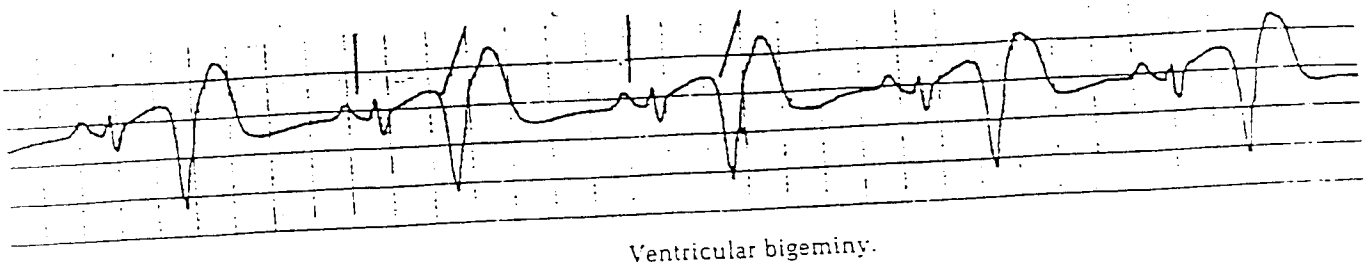
(1) Different shapes, multiform or polymorphic

(2) Different ectopic foci

(3) Different route through myocardium



c. Bigeminal PVCs - every other beat is a PVC



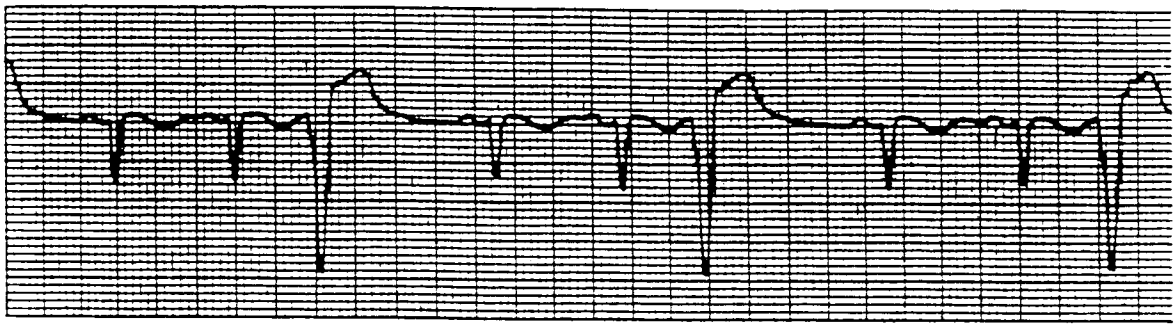
Ventricular bigeminy.

NOTE: Rule of bigeminy: A long cycle tends to precipitate a PVC after the next supra-ventricular beat, implying a long cycle causes a long refractory period.

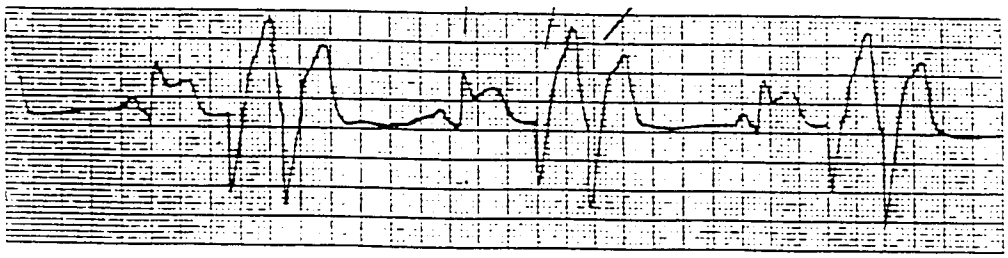
d. Trigeminal PVCs

(1) One PVC for 2 normal beats

(2) Two PVCs for 1 normal beat

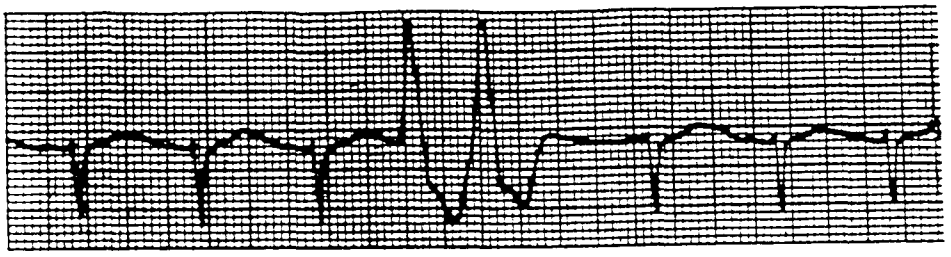


Ventricular trigeminy—two normal complexes and one PVC.



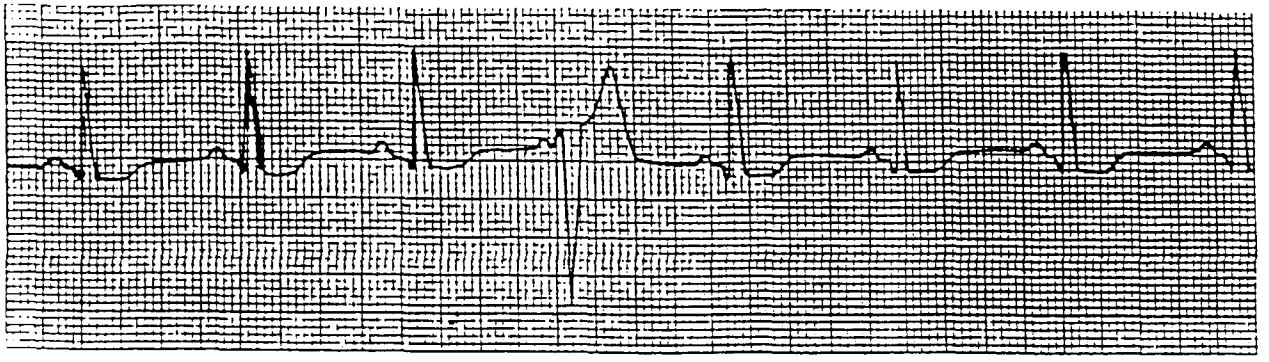
Ventricular trigeminy—one normal complex and two PVCs.

e. Paired PVCs - 2 in a row or sequential



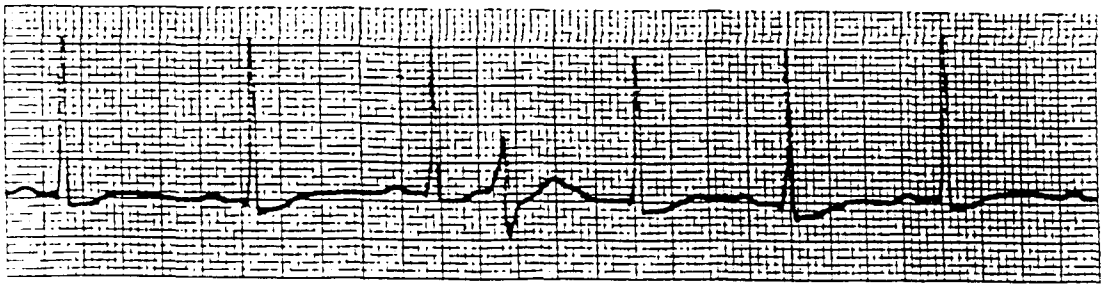
Paired PVCs.

f. End diastolic - occur late in the cycle before ventricles completely activated by sinus beat



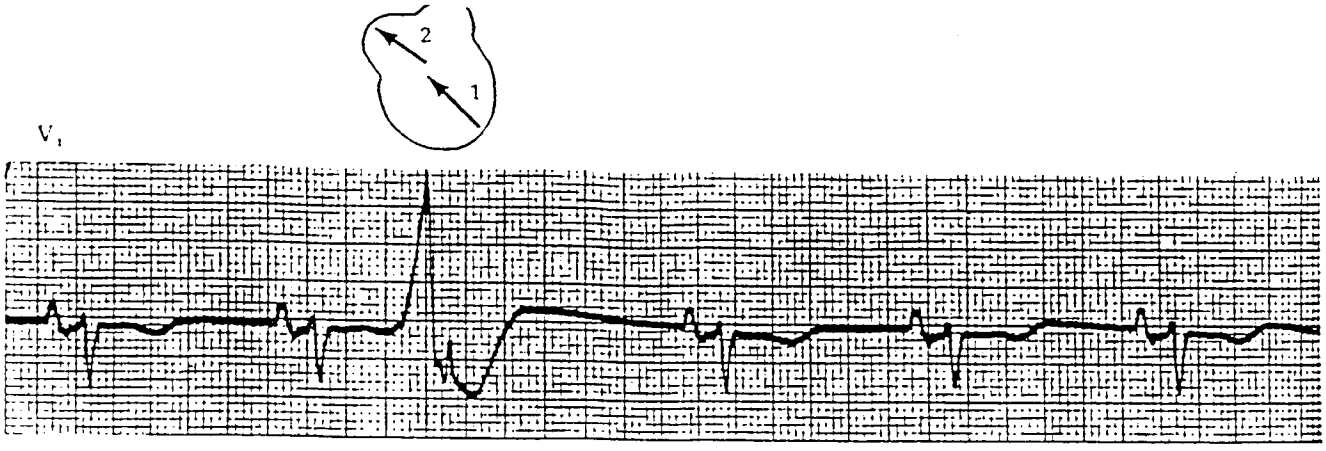
. End-diastolic PVC.

g. Interpolated - sandwiched in between 2 normal sinus conducted beats. Does not have full compensatory pause.



(1) PVC' s with retrograde conduction (50% of them)

(2) P wave following PVC is conducted into ventricle



PVC with retrograde conduction to the atria.

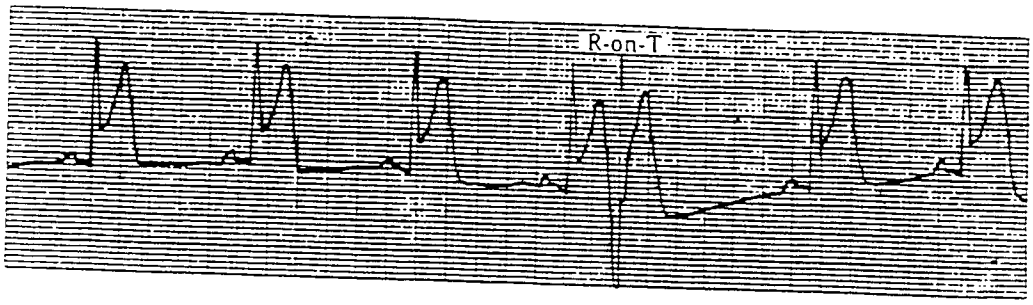
h. Fascicular - may be narrower than other PVC's

i. R on T - PVC lands within T wave

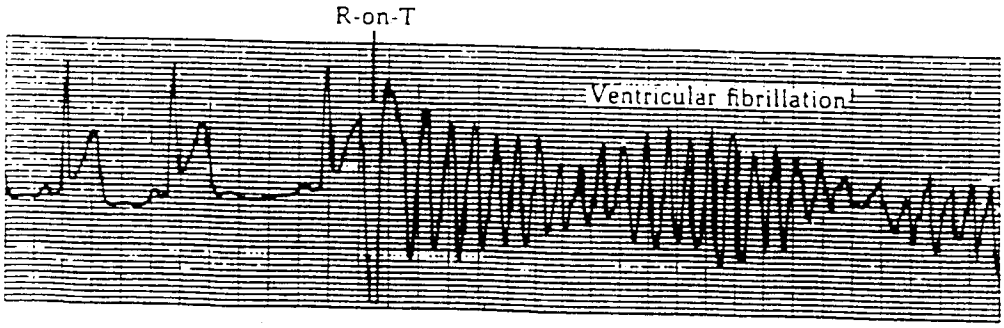
(1) Unstable myocardium

(2) Considered more dangerous in acute MI

(3) More likely to precipitate ventricular fibrillation



R-on-T phenomenon.



R-on-T phenomenon causing venticular fibrillation.

8. Treatment

   a. In face of acute MI - likely to treat more aggressively

      (1) Lidocaine

      (2) Pronestyl

      (3) Bretylium

   b. Correct underlying contributing factors

      (1) Electrolyte imbalance

      (2) Hypoxia

      (3) Acidosis

   c. Other antidysrhythmias

      (1) Quinidine

      (2) Betablockers: Inderal; Breviblock; others

9. Nursing interventions

   a. Identify and document the rhythm

   b. Assess patient for signs/symptoms, ↓ CO, ↓ tissue perfusion

      (1) VS, system assessment

      (2) Mentation

   c. Notify MD of changes

   d. Assess for underlying causative factors (collaborative)

      (1) Electrolyte imbalances

      (2) Hypoxia

      (3) Acidosis

      (4) Other

e. Antidysrhythmics may be indicated

(l) Lidocaine - IV for use in acute MI

(2) Pronestyl - IV or PO - usually second line drug

(3) Quinidine - PO; sometimes (rarely) IM; never IV

(4) Betablockers - IV or PO gaining popularity

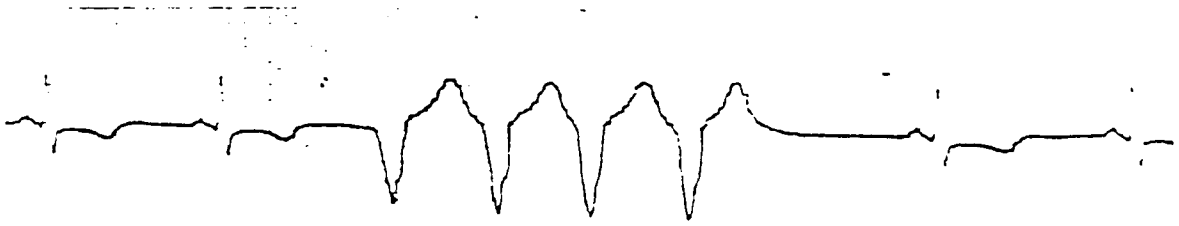
f. Monitor and document patient response to treatments

D. Ventricular Tachycardia (VT). At least three consecutive PVC's with rate of more than 100 bpm

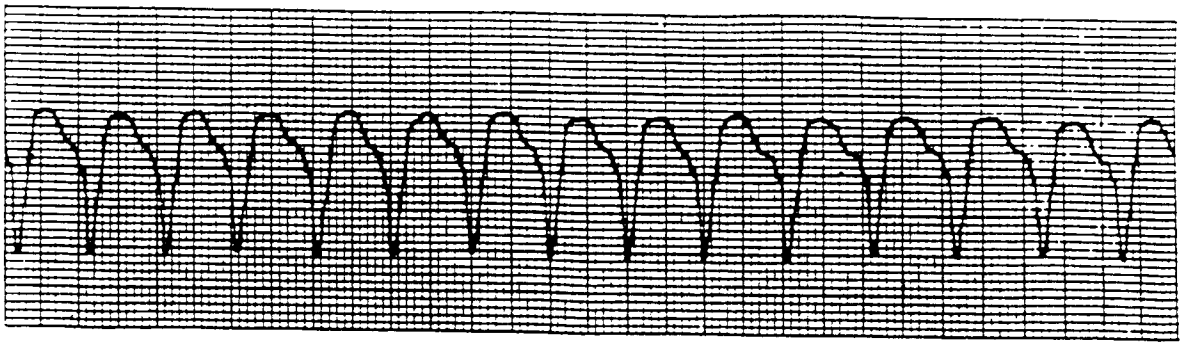
1. Pediatrics: Seen in many clinical situations in children such as surgery, tumors.

2. Differentiate between nonsustained and sustained V-Tach

a. Nonsustained VT: terminates spontaneously within 30 seconds



b. Sustained VT: Consecutive ventricular extrasystoles at a rate greater than 100 beats/minute, sustained longer than 30 seconds. If less than 30 seconds requires intervention for hemodynamic instability.



2. Mechanism

   a. Reentry (likely post MI)

   b. Triggered activity (likely digitalis toxicity or excess catecholamines)

3. Causes: Similar to those of PVC's

4. Clinical features - sudden onset

   a. Nonsustained VT

      (1) May be seen with or without heart disease

      (2) Symptoms range

         (a) Palpitations

         (b) Dizziness, lightheadedness

         (c) Syncope or recurrent syncope

      (3) In patients with severe LV dysfunction it is thought to be a precursor of sustained VT or VF

   b. Sustained VT: Usually more severe symptoms

      (1) Patients with 1 episode of sustained VT after acute MI may do okay after acute phase

      (2) Patients with recurrent sustained VT, that is refractory to drugs in the face of a large anterior MI with CHF may have a one year mortality as high as 80%

5. Significance

   a. Depends upon duration

   b. Can cause serious hemodynamic consequences

   c. Can suddenly change to ventricular fibrillation

6. ECG characteristics for differential diagnosis of wide QRS tachycardias

NOTE: Electrophysiologic studies confirm certain surface ECG clues that allow 90% accuracy in the diagnosis of wide QRS tachycardias.

a. Traditional criteria for evaluation of ventricular tachycardia

(1) Hemodynamic criteria - does not correlate to rhythm. VT may be well tolerated

(2) Age - cannot be used

(3) Heart rate - cannot be used

(4) QRS axis

(a) "No man's land" axis (-90 to -l80 degrees) favors VT

(b) Otherwise, helpful only if QRS is upright in V_1 - then both abnormal left axis deviation and abnormal right axis deviation favor VT

NOTE: QRS axis is of no specific help if QRS is negative in V_1.

(5) QRS with: > 0.l4 second favors VT

NOTE: There are exceptions to the QRS width:

(a) Pre-existent BBB

(b) Antidromic circus movement tachycardia

(c) Atrial fibrillation with conduction over an pathway and

(d) Digitalis toxicity

(6) Presence of fusion beats still indicate VT

(7) AV dissociation (lack of l:l A-V conduction) confirms VT; cannon A waves in jugular pulse, varying intensity of S_1, changing SBP

(8) Precordial concordance - indicates VT

b. New steps in differential diagnosis of wide QRS tachycardia

(1) Leads of choice

(a) I and aVF - axis

(b) V_1, V_2, V_6 - morphologic clues for ventricular ectopy

(2) QRS configuration

(a) When QRS configuration is mainly positive in V_1

1) Suspect ventricular tachycardia if

a) Monophasic complex

b) Diphasic complex

c) If there is precordial concordance and V_1 is positive, check V_6 - if the S wave is deeper than the R wave is tall it's VT

d) Rabbit ear clue - described by Gozensky and Thorne (two nurses) 1970

1 If two positive peaks in V_1, and the left is taller than the right peak, it favors ventricular ectopy (17:1)

2 If right peak is taller than the left is not diagnostic, as this finding is common to both

2) Suspect supraventricular tachycardia (SVT) if

a) Triphasic QRS in V_1

b) If triphasic in V_6, SVT
If V_1 is positive, q in V_6 SVT
If V_1 is negative, q in V_6 VT

(b) When QRS is mainly negative in V_1

1) Suspect ventricular tachycardia if

a) A broad (fat) R wave of 0.03 seconds or more in V_1 or V_2

b) A notched or blurred downstroke on the S wave or QS wave in V_1 or V_2

c) A delayed nadir - that is a distance of 0.07 seconds or more from the onset of the ventricular complex to the nadir of the QS or S in V_1 or V_2

d) Any Q in V_6 but only if the complex is mainly negative in V_1

2) Suspect supraventricular tachycardia if

a) LBBB aberration in Vl and V2 with narrow r wave

b) Clean, swift downstroke of S wave in V_1 and V_2

c) V_6 = monophasic R complements criteria seen in V_1 and V_2 but is not diagnostic

c. Bedside diagnosis of VT

(1) Physical signs

(a) Irregular cannon a waves in jugular pulse

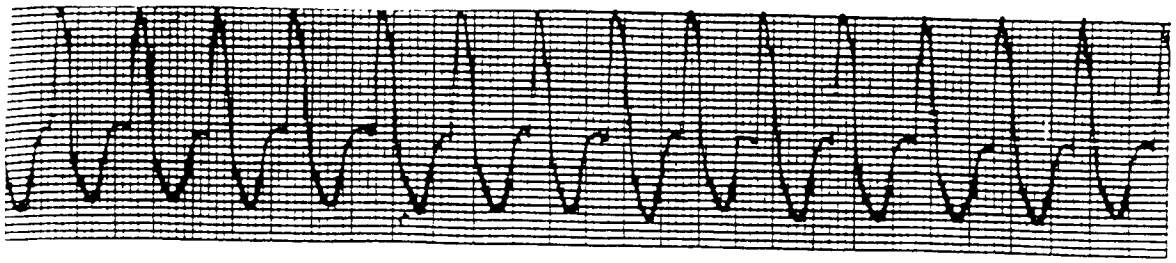
(b) Varyng intensity of S_1

(c) Beat to beat changes in SBP (changing Kartokoff sounds)

(2) History (if yes to both questions - suspect VT)

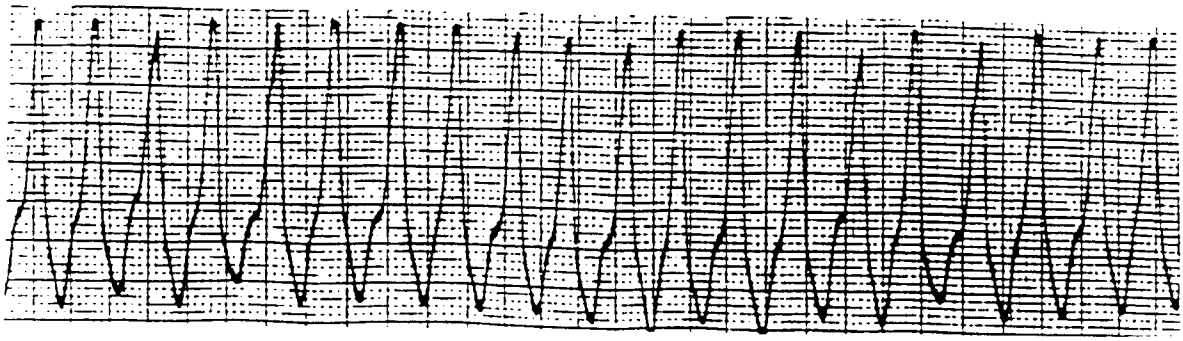
(a) Prior MI

(b) Symptoms of tachycardia only after MI

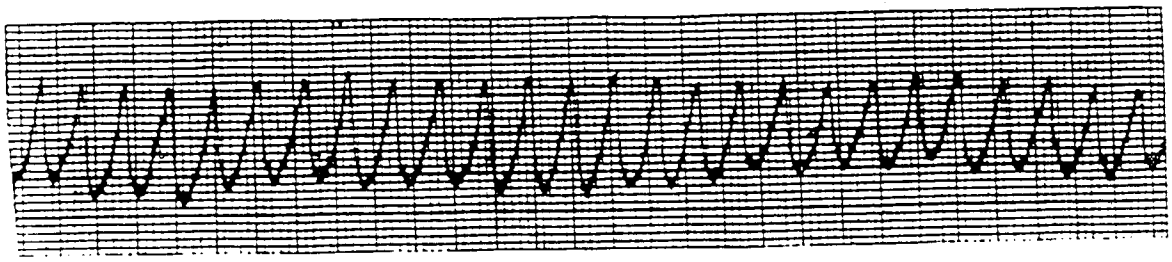


Rapid **Ventricular Tachycardia**

Ventricular tachycardia.



Extreme Ventricular Tachycardia (Ventricular Flutter)



7. Treatment of ventricular tachycardia

   a. ACLS (SeeAlgorithm)

      (1) If patient has pulse and is awake and VS okay - Lidocaine per SOP may be used

      (2) If pulse, but hemodynamically compromised

         (a) If patient unconscious - emergency cardioversion

         (b) If patient awake, consider sedation prior to cardioversion

      (3) Pulseless - treat as V fib and defibrillate

   b. When doubt

      (1) If hemodynamic compromise - ACLS

         (a) Pulseless - defibrillate

         (b) Pulse present - cardiovert

      (2) Otherwise be methodical in differentiating aberration from ectopy

      (3) If still in doubt - Pronestyl rather than Verapamil is the drug of choice

         (a) Hemodynamic deterioration after IV Verapamil is well documented - necessitates immediate cardioversion

NOTE: Verapamil slows SA and AV nodal conduction and prolongs refractory time. It decreases contractility, afterload, arterial pressures, vascular tone and O_2 demand. But, if the AV node is the area of delay for accessory path circuit rhythm, results of Verapamil are disasterous.

(b) Ensuing hypotension after Verapamil may render cardioversion unsuccessful

(c) Pronestyl has therapeutic action for both VT and SVT

1) Antifibrillatory

2) Terminates VT

3) Slows conduction in accessory pathway and the retrograde fast AV nodal pathway (useful for WPW and AV nodal reentry)

c. Electrical therapy - antitachycardia pacing for slower VTs

8. Nursing concerns

a. Identify and document ventricular tachycardia

b. Assess patient

c. Tell patient to cough

d. Notify MD

e. ACLS

E. Ventricular Fibrillation - the most common cause of sudden death

1. Electrical chaos and mechanical failure - a disorganized quivering of the heart

2. Mechanism: may be initiated by

a. Altered automaticity

b. Reentry

c. Triggered activity

3. Causes

a. Coronary artery disease and sequelae

b. Similar to PVCs

4. Clinical implications

   a. Pulseless, nonbreathing, sudden death

   b. Initiate BLS/ACLS immediately for survival (See M-2 for ACLS Algorhythm)

   c. In the presence of ischemia, primary V fib has a high recurrence rate

   d. Prompt early treatment of VF,

      (1) Is usually successful if VF drug induced, or during cardiac catheterization or acute MI before onset of shock or cardiogenic failure

      (2) In the face of shock or LV failure

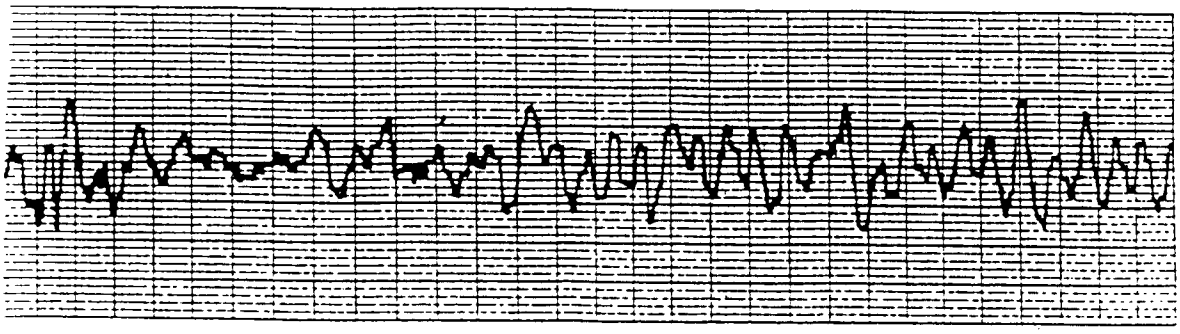
         (a) Treatment is only 30% successful

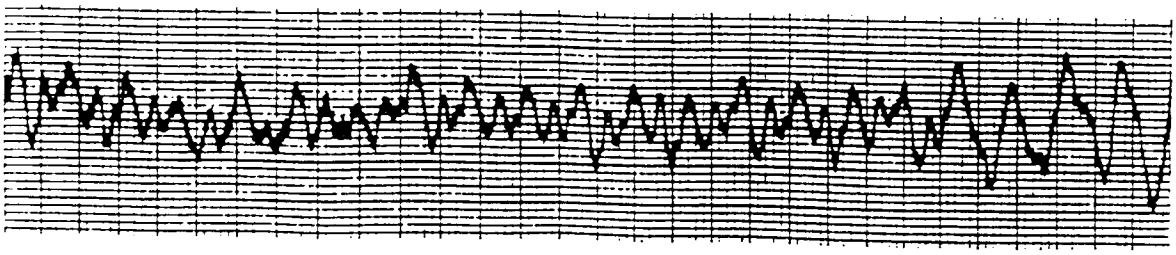
         (b) Recurrence is common

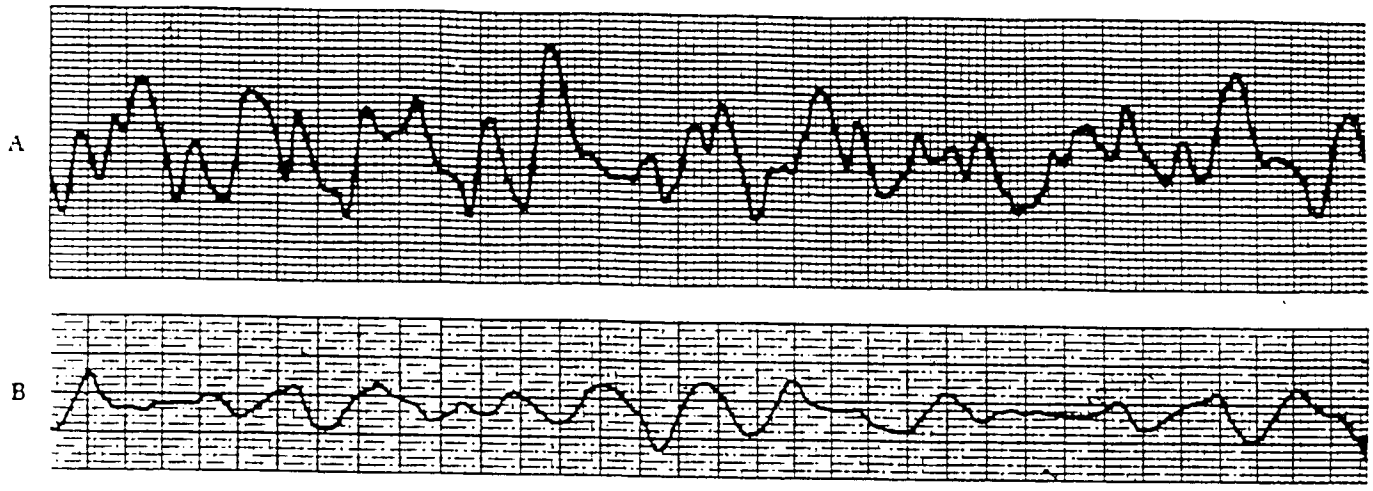
         (c) Prognosis is poor

      (3) VF occurring more than 48 hrs after MI commonly recur and has a poor prognosis

5. ECG recognition - erratic electrical activity with irregular nonuniform lines







Ventricular fibrillation. A, With a coarse fibrillatory line. B, With a fine fibrillatory line.

6. Treatment

   a. Immediate recognition

   b. DC defibrillation of VF - may be followed by drug therapy

      (1) Lidocaine

      (2) Pronestyl

      (3) Bretylol/Bretylium

   c. Survival chances are higher if

      (1) Occurs less than 24 hrs after onset of MI symptoms

      (2) Fewer than 4 shocks are required to terminate VF

      (3) No prior antidysrhythmic drugs were used

      (4) Sinus or paced rhythm, or atrial fibrillation is the lst rhythm within 1 minute following defibrillation

   d. Long term treatment

      (1) Oral antidysrhythmics

      (2) AICD (Automatic implantable cardioverter-defibrillator)